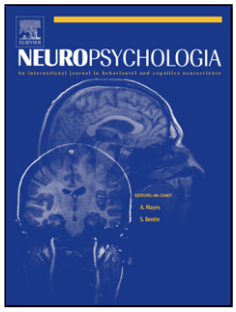

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PII: S0028-3932(12)00063-2
DOI: doi:10.1016/j.neuropsychologia.2012.02.003
Reference: NSY 4401

To appear in: *Neuropsychologia*

Received date: 22-6-2011
Revised date: 20-1-2012
Accepted date: 4-2-2012

Please cite this article as: Broyd, S. J., Richards, H. J., Helps, S. K., Chronaki, G., Bamford, S., & Sonuga-Barke, E. J. S., Electrophysiological markers of the motivational salience of delay imposition and escape, *Neuropsychologia* (2010), doi:10.1016/j.neuropsychologia.2012.02.003

Electrophysiological markers of the motivational salience of delay imposition and escape

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Abstract

Background: The ubiquitous tendency to choose immediate over delayed rewards can, in extremis, lead to maladaptive preferences for smaller sooner over larger later rewards (i.e., impulsive choice) in certain pathological groups. **The delay aversion hypothesis provides one possible account of impulsive choice and argues that this tendency is motivated by the avoidance of the negative affective states associated with delay imposed prior to the delivery of a large reward.** This model also predicts that on non-choice tasks individuals will be motivated to work harder and more efficiently, when given the opportunity to avoid delay. In the current paper we studied the neural markers of the motivational salience of the imposition and escape from delay using a simple reaction time task under two conditions: First where fast responses were expected to lead to delay escape and second where delay was inescapable.

Methods: Forty participants performed the Escape Delay Incentive (EDI) task during which they were asked to respond as quickly as they could to a target stimulus. The EDI task included two conditions: first, a *Delay Escape* condition where fast responses led to the avoidance of delay and a *Delay No-Escape* condition in which a delay was presented on every trial irrespective of response speed. EEG was recorded from 66 equidistant electrode sites across the scalp. The neural response in these two conditions was compared in terms of contingent negative variation (CNV; preparation of motivated responses) and Late Positive Potential, LPP; evaluation of performance feedback).

Results: As predicted individuals responded more quickly and showed enhanced CNV amplitude to *Delay Escape* compared with *Delay No-Escape* trials. Enhanced LPP amplitude was also observed when participants were not able to avoid the delay in the *Delay Escape* condition. ADHD symptoms were associated with larger CNV differences between *Delay Escape* and *Delay No-Escape* conditions. **An association between ADHD symptoms and the LPP in the Delay Escape condition did not reach significance.**

Conclusion: The results of the current study suggest that delay escape is a potent reinforcer at both behavioural and neural levels. Future research should extend this analysis to clinical samples using a broader range of delays and across imaging modalities.

Key words: Delay Aversion, ADHD, event-related potential, CNV, LPP

Research highlights:

- Escape from delay reinforced shorter and less variable response times in an Escape from Delay Incentive (EDI) task
- CNV amplitude was enhanced following *Delay Escape* compared with *Delay No-Escape* cues.
- The CNV difference between *Delay Escape* and *Delay No-Escape* conditions was associated with ADHD symptoms.
- Finally, LPP amplitude was enhanced when participants failed to escape delay in the *Delay Escape* condition.
- These findings suggest delay itself may be a potent reinforcer and highlights the potential role for delay in inter-temporal choice.

Introduction

In our daily lives, we are frequently forced to choose between alternatives which differ in terms of their nature, size and timing. With respect to the timing, both humans and non-human animals have a tendency to choose immediate over delayed outcomes when all else is considered equal (for a comprehensive review see Luhmann, 2009). In certain situations this tendency can lead to maladaptive performance when rewards of lesser absolute value delivered sooner are chosen over more valuable rewards delivered after a delay; i.e., impulsive choice (Duva, et al., 2011; Simon, et al., 2007). The tendency towards impulsive choice varies between individuals (e.g., Kirby & Marakovic, 1996; Shamosh & Gray, 2008). It is stronger in certain personality types (e.g., Hirsh, et al., 2010) and is associated with certain psychopathologies (e.g., Bobova, et al., 2009). Patients with attention deficit/hyperactivity disorder (ADHD) are more impulsive in this sense (Barkley et al., 2001), as are patients with schizophrenia (Heerey, et al., 2007); cocaine-addicts (Kirby & Petry, 2004), pathological gamblers (Dixon, et al., 2003), smokers (Businelle, et al., 2010) and alcoholics (Mitchell, et al., 2005; Petry, 2001); while the opposite seems to be the case for individuals with higher levels of intelligence (Shamosh & Gray, 2008).

A number of models have been proposed to explain the disproportionate power of immediate rewards and their influence on choice (Ainslie, 1975; Marco, et al., 2009; Sagvolden, et al., 2005; Sonuga-Barke, 2002; Sonuga-Barke, et al., 2010; Tripp & Wickens, 2008). The two most well established models regard impulsive choice as either evidence of (i) temporal discounting of rewards as they are moved through time into the future (TD, Ainslie, 1975; Kahneman & Tversky, 1979) or (ii) the failure of higher-order executive-based inhibitory control whereby an individual is unable to suppress the drive to respond to the immediate option (Barkley et al., 2001; Cardinal, et al., 2001; Marco et al., 2009). From a temporal discounting perspective the choice of the smaller sooner reward over the larger later reward occurs because temporal discounting typically follows a hyperbolic or quasi-hyperbolic function leading to a preference reversal with increasing delay (Ainslie, 2005). According to this model, more impulsive individuals discount the future at a higher rate (e.g. Barkley, et al., 2001). In contrast, from the executive function perspective impulsive individuals are predicted to have reduced executive-related inhibitory control – an effect that can be observed in addiction (Bickel et al., in press) and other impulsive behaviours where executive dysfunction is also observed (e.g. choice impulsivity in schizophrenia, Heerey et al., 2007). At the neurobiological level the two models make somewhat different predictions. From the temporal discounting perspective, steeper discounting has been linked to attenuated dopamine signalling of delayed rewards in the ventral striatum which may be due to the reinforcing properties of primary reinforcers failing to transfer to secondary reinforcers (Kobayashi & Schultz, 2008), a process that normally occurs during reinforcement learning (see Cardinal 2006 for a review). Further, immediate reward preference is found to correlate with activation of ventral striatum, medial orbitofrontal cortex and medial prefrontal cortex (Hariri, et al., 2006; McClure, et al., 2007; McClure, et al., 2004; Tanaka, et al., 2004; Wittmann, et al., 2007). Evidence linking the disruption of executive brain circuits and impulsive choice has also been found. Activity within frontostriatal brain circuits mediates key executive control regions including dorsolateral prefrontal and dorsal striatal areas with strong connections to other cortical regions including the parietal cortex (McGuire & Botvinick, 2010; Rubia, et al., 2006; Savine & Braver, 2010). Disrupted activation within these networks is thought to underpin the inability to wait for larger delayed rewards in a number of psychopathological groups (see Carter 2010 for a review). Furthermore, larger later choices are found to activate lateral prefrontal and parietal areas (McClure et al., 2004), bilateral posterior insula cortex (Wittmann et al., 2007) and posterior cingulate cortex (Luhmann, et al., 2008; Weber & Huettel, 2008).

The delay aversion model offers a different perspective on impulsive choice from these two accounts. It is based on the idea that the imposition of delay creates a negative emotional state which generates a motivation to avoid delay and escape its aversive character where this is possible. Impulsive choice is therefore reinforced where the choice of immediate over delayed alternatives reduces the level of delay (Sonuga-Barke et al., 2010). To date, the

neurobiological basis of these delay aversion behaviours has not been directly examined. However, a number of predictions are possible. According to the delay aversion model, cues signalling inescapable delay should activate those regions of the brain previously shown to respond to upcoming aversive events (e.g., amygdala and insula). Consistent with this notion, Plitcha et al. (2009) found enhanced amygdala activation to a delayed reward choice option in patients with ADHD. Furthermore Sonuga-Barke et al. (2011) reported an association between impulsive choice of immediate rewards and a functional variant of the serotonin transporter genotype known to affect amygdala function . To an equal and opposite degree, cues signalling the opportunity to escape delay **(in a similar way to other incentive-predicting cues)** would be predicted to activate reward centres of the brain (e.g., ventral striatum; Gregorios-Pippas et al., 2009). This account can be extended to non-choice tasks where delay imposition and delay escape should impact upon levels of task-related motivation. Where escape from delay is made contingent on hard work and/or good performance individuals should work harder or perform better to avoid the aversive properties of delay. Similarly, cues predicting a performance-delay contingency during such tasks would be predicted to activate those regions of the brain engaged during response preparation.

In the current study we used a new paradigm combined with electrophysiological measurement to investigate the neural markers of the motivational salience of performance-contingent delay imposition and delay escape. The *Escape from Delay Incentive* (EDI) task allows for the comparison of brain activity on trials during which participants are given the opportunity to avoid post-response delay through fast responding on a simple reaction time (RT) task (*Delay Escape* condition) and those where delay was always imposed after responses irrespective of performance (*Delay No-Escape*). We made a number of predictions. First, at a behavioural level, we predicted that cues predicting the opportunity to escape delay would reinforce faster and less variable RTs. **Second, at the neural level we predicted that such cues would also elicit enhanced anticipatory brain activity as individuals prepare to respond quickly.** To test this prediction we focused on the contingent negative variation (CNV), a slow negative brain potential elicited by an informative cue which signals the impending onset of an imperative stimulus requiring the participants attention and response (Walter, et al., 1964). The CNV, which is maximal over frontocentral sites, reflects anticipatory attention and effortful processing and is comprised of two sub-components, an early wave ('orienting' O wave) related to the alerting properties of the warning stimulus and a later component ('expectancy' E wave) which is associated with anticipation and the preparation of a motor response (Brunia, et al., 2011; Van Boxtel & Bocker, 2004), as well as motivation (Cant & Bickford, 1967; Irwin, et al., 1966). Despite its role in anticipatory attention, the CNV has received relatively little attention in terms of its role in the processing of reinforcement contingencies; although an enhanced CNV in anticipation of affective or motivationally salient stimuli such as interesting or threatening stimuli has been reported (Baas, et al., 2002; Böcker, et al., 2001; Klorman & Ryan, 1980). We also extended our investigation to look at neural responses to performance-related outcomes (i.e., whether participants were successful in avoiding delay or not on *Delay Escape* trials). Several ERP components are known to be sensitive to the affective content of a reinforcing stimulus. For example, the P300, a positive component which emerges between 200-600 milliseconds over centroparietal sites, is modulated by the emotional salience of a stimulus (Hajcak & Olvert, 2008), and is enhanced to rewarding outcomes (i.e. win vs. loss, Wu & Zhou, 2009; Yeung & Sanfey, 2004). The P3 component elicited by informative feedback (e.g. reinforcement) is more commonly referred to as the Late Positive Potential (LPP). This component is consistently enhanced to pleasant and unpleasant pictures and words relative to neutral stimuli (for a review see Hajcak, et al., 2010), and larger for more arousing or intense outcomes (Schupp, et al., 2000). In children with ADHD, the LPP is enhanced to omitted rewards (Van Meel, et al., in press). The underlying sources of the LPP are likely to be in occipital and parietal cortices (Keil, et al., 2002; Sabatinelli, et al., 2007), which receive projections from the amygdala (Bradley, et al., 2003) - consistent with the notion that this component is implicated in the processing of affective visual stimuli. In the current study, we predict that because the

imposition of delay (when the opportunity to escape is possible) is aversive, the LPP would be enhanced on *Delay Escape* trials where individuals failed to escape delay compared with trials where delay escape occurred.

In order to test the generalisability of our findings, our sample covered a broad range of ages across adolescence and early adulthood. While we expect the predicted effects to be seen in both the adolescent and young adult samples, adolescence is thought to be an influential period in relation to the development of reward-related brain circuitry (Galvan, 2010; Geier, et al., 2010). Although initial neuroimaging work suggested that adolescents exhibit hypo-responsive activation in the ventral striatum during reward anticipation (Bjork, et al., 2004), further studies typically report that adolescents display hyper-responsive activation to rewards (e.g. Casey et. al., 2008; Somerville et. al., 2010). Such alterations may result in increased reward-seeking behaviour and underpin the patterns of exaggerated risk-taking and impulsive choice which often characterise adolescence (Fareri, et al., 2008; Somerville, et al., 2010). While keeping this possibility in mind, the current paper was not able to test this hypothesis definitively because of a confound between age group and gender Finally, in our sample we examined the association between self-report ADHD symptoms and the reinforcing effect of escape from delay on behavioural and brain markers. **In keeping with the notion that individuals with higher ADHD symptoms would find escape from delay more reinforcing and its imposition more punishing than individuals with fewer ADHD symptoms, we predicted that higher ADHD symptom scores would be associated with increased anticipatory brain potentials (CNV) following cues on *Delay Escape* compared with *Delay No-escape* trials, as well as an increased LPP following failures to escape delay on *Delay Escape* trials.**

Materials and Methods

Participants

Eighteen undergraduate students (2 male, mean age: 21.8 years, SD: 4.5 years) and 22 adolescents (all male, age: 15.0 years, SD: 0.7 years) participated for course credit and/or small monetary incentive. Two participants in the adolescent sample who had a clinical diagnosis of ADHD were recruited from local Hampshire clinics and twenty healthy adolescents were recruited from local secondary schools. Participants had not taken any medication for at least 24 hours prior to the experiment, the two adolescents with clinical diagnoses discontinued their methylphenidate medication for 24 hours prior to the experiment, and all participants had normal or corrected vision and hearing. The groups did not differ in ADHD symptoms (young adults M = 11.9, male adolescents M = 16.8, $F(1,35) = 2.38$, $p = .131$, *ns*). **Although we refer to these groups as adolescent and young adult samples, please note the groups also differ in gender (22 males in the adolescent group and 2 males in the young adult group). This limitation is addressed further in the discussion.** The research protocol was approved by the University of Southampton, School of Psychology Ethics Committee and the Southampton and South West Hampshire Research Ethics Committee B.

Experimental Paradigm and Procedure

All participants were familiarised with the EEG recording procedure before informed consent was taken. Participants completed a short screening questionnaire to assess vision problems, medication and psychotropic substance use, and neurological disorders. They were then fitted with recording electrodes and seated in a comfortable chair approximately 80 cm away from the monitor in a darkened room. Participants took part in two core assessments: a monetary incentive task and the EDI task. Undergraduate participants also completed an approach/avoidance task. The approach-avoidance and monetary incentive tasks were completed as part of another experiment and will not be reported here. The order of the tasks was counter-balanced and participants were

encouraged to take a short break between each task block and a longer break between each experimental task. Participants were instructed to sit as still as possible and to minimise blinking.

At the start of each trial in the EDI task, participants were presented with one of two blue cue stimuli which was followed shortly afterwards by the target stimulus, a white square. Participants were instructed to respond to the target (a white square) as quickly as possible with the thumb on their dominant hand via a button box key. Feedback was provided; a green tick signalled that their response was 'fast enough' while a red cross signalled that their response was 'too slow'. This task included an algorithm which tracked each participant's response on a trial by trial basis and adjusted the response window for a 'fast enough' response so that all participants received positive feedback, based on their own performance, on 66% of trials **(success rate 0.67 for all participants).** There were two trial types in each session which were presented with equal probability and in random order: *Delay No-Escape* and *Delay Escape*. In the *Delay No-Escape* condition (signalled by the blue circle cue) participants were told that although they would receive feedback about the speed of their response (a fast response would receive positive feedback and a slow response would receive negative feedback), they would experience a delay period after their response on every trial irrespective of performance. In the *Delay Escape* condition (signalled by a blue octagon cue) participants were informed that a fast response would receive positive feedback and allow them to progress directly to the next trial and avoid the post response delay, while a slow response would receive negative feedback and be followed by delay. The delay period was signalled by a bar presented centrally on the computer monitor that disappeared a little at a time. The delay stimulus appeared on screen for a variable duration of between 8 and 10 seconds with a 1 second inter-trial interval. Two practice blocks were completed prior to the experimental blocks to allow participants to learn the association between each cue and experimental condition. The first practice block (10 trials) included *Delay No-Escape* trials only. The second practice block (20 trials) included both *Delay Escape* and *Delay No-Escape* trials. The cue and target stimuli were each presented for 250 ms and for the adult sample were separated by a fixed 2000 ms inter-stimulus interval (ISI). For the adolescent sample, the ISI was varied randomly between 2000 and 2500 ms (M: 2250 ms). The feedback stimuli were presented for 1500 ms and appeared on screen 1450 ms following the offset of the target stimulus. Young adult participants completed 4 experimental blocks of 40 trials; adolescent participants completed 4 experimental blocks of 30 trials.

To examine the relationship between the motivational salience of 'delay escape' and ADHD, all young adult participants were asked to complete the Adult ADHD Rating Scale (Barkley & Murphy, 1998), and the parents of the adolescent participants completed the Child ADHD Rating version of this scale (DuPaul, et al., 1998). This scale contains 18 symptom items for ADHD as per the DSM-IV, and includes two related subscales of inattention and hyperactivity/impulsivity. This scale has good construct validity and test-retest reliability (construct validity .35-.85, 4 week test-retest reliability .78-.86, for a review see Collet, et al., 2003).

Electrophysiological Acquisition and processing

An electrode cap (Easycap, Herrsching, Germany) containing 66 equidistantly spaced silver/silver chloride (Ag/AgCl) electrodes was fitted to each participant and EEG data was recorded using Neuroscan Synamps^2 70 channel EEG system, DC-coupled recording equipment. The data were sampled at 500 Hz with a low pass filter at 70 Hz and referenced to an electrode on the nose. A ground electrode was fitted midway between the electrode at the vertex and frontal sites. Vertical electro-oculogram (vEOG) was recorded from four electrodes: two bipolar electrodes were placed directly beneath the left and right eyes and affixed with tape, while the two electrodes placed above the right and left eye were included within the electrode cap. Impedances for vEOG, reference and cap electrodes were kept below 5 kΩ. On each trial ERP data were examined to the cue and feedback stimuli (positive vs. negative). The continuous EEG data were transformed using a DC offset and linear detrend algorithm. The ERP epoch to the cue began 200 ms prior to stimulus onset and ended 2000 ms post-stimulus presentation. The ERP epoch to the feedback

stimulus also included 200 ms pre-stimulus activity and extended 1500 ms post-stimulus. An ocular artifact reduction procedure (Semlitsch, et al., 1986) based on left eye vEOG activity was used to remove blink artifacts and other eye-movements from the ERP data. Further, any epoch that exceeded ± 100 μV at any non-frontal scalp site was rejected. ERP data to all stimuli were baselined to the 200 ms pre-stimulus interval and filtered 48 dB down at 32 Hz using a low-pass filter for analysis. Individual ERP averages were based on a minimum of 20 trials with the exception of negative feedback trials in which a criterion of a minimum of 10 trials was used. Based on these criteria, data from one participant in the adolescent group were excluded for ERP analyses to the cue and data from 3 young adults and 5 adolescents was removed from the ERP analyses to the feedback stimuli. The number of trials used to estimate the CNV was: young adults escape trial M = 66.8 SD = 12.3, no escape trial M = 65.4 SD = 12.7; adolescents escape trial M = 41.3 SD = 9.8, no escape trial M = 39.2 SD = 9.4. The number of trials used to estimate the LPP during the *Delay Escape* trials was: young adults: Positive Feedback M = 46.0 SD = 7.8, Negative Feedback M = 20.7 SD = 5.9; adolescents: Positive Feedback M = 32.5 SD = 7.2, Negative Feedback M = 18.3 SD = 3.5. The number of trials used to estimate the LPP during the *Delay No-Escape* trials was: young adults: Positive Feedback M = 43.0 SD = 10.5, Negative Feedback M = 20.4 SD = 4.4; adolescents: Positive Feedback M = 33.3 SD = 8.0, Negative Feedback M = 15.5 SD = 2.5. Following the cue stimulus a clear CNV emerged around 600 ms. Mean amplitudes for the CNV was quantified in two time windows (young adults: CNV1: 600 to 1100 ms and CNV2: 1100 to 1600 ms; adolescents: CNV1: 650 to 1150 ms and CNV2: 1150 to 1650 ms). Following the onset of the feedback stimulus, the LPP component emerged around 300 ms and was largest at occipital sites. As this component extended from around 300 ms until 600 ms post-stimulus, we analysed the data in two time segments, an early LPP component (LPP1; young adult: 320 to 450 ms and adolescent: 290 to 450 ms) and a later LPP component (LPP2, young adult and adolescent: 450 to 600 ms).

Data analysis

Performance data: To examine the effects of delay escape on performance we compared mean (MRT) and standard deviation of reaction time data (SD of RT) in the *Delay Escape* and *Delay No-Escape* conditions. RT data were trimmed to remove responses which were faster than 150 milliseconds and exceeded +/- 2.5 SD around the mean response time. MRT and SD of RT were analysed using repeated measures ANOVA with condition (Delay No-Escape, Delay-Escape) as the within-subjects factor and Group as the between-subject factor.

ERP data: ERP data were analysed using Scan 4.4 (www.neuroscan.com), the cue and feedback stimuli were examined separately and the mean amplitude data in the *Delay Escape* condition with the *Delay No-Escape* conditions compared. ERP data were examined for outliers (+/- 2.5 SD around the mean). All analyses were repeated with and without any identified outliers and where the pattern of effects remained the same, ERP data were retained in original form. ERP data were examined at selected sites, where the chosen ERP components were maximal. Consistent with previous ERP literature, the cue-locked CNV was largest at frontomedial sites (Brunia et al., 2011). Therefore statistical analyses for the CNV included a laterality factor which was the average of frontocentral electrodes in the left hemisphere (electrodes 7 and 18), midline (electrodes 1 and 2) and right hemisphere (electrodes 3 and 8; see Figure 1). Further, as CNV mean amplitude was examined in two time windows, CNV1 and CNV2, we included these levels within a time-window factor in our analysis. Our analysis of the feedback-related LPP component also included a time window factor with two levels: LPP1 and LPP2. Consistent with previous literature, the LPP component was most pronounced at parietal-occipital sites (Hajcak et al., 2010). Therefore statistical analyses for the LPP component included a sagittal factor which incorporated electrodes at central, cento-parietal and parietal/occipital sites (electrodes 1, 5, 13, 25).

Cue CNV data were entered into a 2 (time window – CNV1, CNV2) by 2 (condition - Delay escape and Delay No-escape) by 3 (laterality - left hemisphere, midline, right hemisphere) by 2 (group – young adults and adolescents) repeated measures ANOVA. As we were especially interested in failed and successful attempts to escape delay, feedback-locked LPP data were examined using a repeated measures ANOVA with time window (LPP1, LPP2) x feedback type (Positive, Negative) x sagittal (1, 5, 13, 25) as within-subjects factors and group (young adults and adolescents) as the between subjects factor for the *Delay Escape* condition only. Where the electrode factor (lateral or sagittal) did not interact with condition, it was dropped from the analyses so that the mean CNV across laterality (left hemisphere, midline, right hemisphere) or the mean LPP across sagittal electrodes (1, 5, 13, 25) were entered into the ANOVA for time window and feedback type. Post-hoc tests were used to follow-up significant main effects and interactions.

Associations between CNV and LPP and ADHD symptoms. Where a main effect of condition on ERP data was identified, we created an Impact of Delay Escape Scores (IDES) for the CNV which was based on the difference in amplitude between Delay No-Escape and Delay Escape trials, and for the LPP we created an Impact of Delay Imposition Index (IDII) which was based on the difference in LPP amplitude between the Delay Escape Failure trials and the Delay Escape Success trials. This was then correlated with self-reported ADHD scores.

Results

Performance data: MRTs were significantly shorter in the *Delay Escape* (MRT - M: 217 ms; SE: 5 ms) compared with the *Delay No-Escape* condition (MRT - M: 232 ms; SE: 7 ms; $F(1,38) = 11.29$, $p = .002$). There was also a tendency for RTs to be less variable in the *Delay Escape* (SD of RT - M: 71 ms, SE: 8 ms) compared with the *Delay No-Escape* condition (SD of RT - M: 87 ms, SE: 9 ms; $F(1,38) = 2.93$, $p = .095$). There was no main effect of group on task performance, nor was group found to interact with condition for either MRT or SD of RT.

Cue CNV: Condition and laterality were not found to interact significantly (p>0.100) so the lateral factor was dropped from the analyses. There was a main effect of condition ($F(1, 37) = 18.96$, $p < .001$): CNV mean amplitude was enhanced to *Delay Escape* (M: -1.08 μV, SE:0.62 μV) compared with *Delay No-Escape* cues (M: 0.98 μV, SE: 0.68 μV; see Figure 1). Condition did not interact with time window ($F(1, 37) = 1.17$, $p = .286$). There was not a main effect of group (F<1), and group was not found to interact with condition ($F(1, 37) = 1.98$, $p = .168$). An effect of time window indicated that mean amplitude was larger in the second (CNV 2; M: -1.24 μV, SE: 0.62 μV) compared with the first time window (CNV1, M: 1.41 μV, SE: 0.62 μV; $F(1, 37) = 62.43$, $p < .001$).

Feedback LPP: Feedback type and time-window were not found to interact significantly with the sagittal factor, so the latter was removed from the analysis (p values>.100). A main effect of feedback type was observed for the LPP ($F(1, 30) = 19.29$, $p < .001$; see Figure 2) and indicated that the mean LPP amplitude was enhanced to negative (M: 16.44 μV, SE: 1.83 μV) compared with positive (10.40 μV, SE: 1.39 μV) feedback. There was an effect of time window with a greater mean amplitude in the first (LPP1, M: 17.38 μV, SE: 1.63 μV) compared with the second time window (LPP2, M: 9.47 μV, SE: 1.67 μV; $F(1, 30) = 28.02$, $p < .001$), however time-window was not found to interact with feedback ($F(1, 30) = 1.64$, $p = .211$). Finally, there was neither a main effect of group nor any interactions between group, feedback or time-window (all F values <1).

Associations between ADHD and ERP correlates: CNV IDES scores (*Delay No-Escape – Delay Escape*) and LPP IDII scores on *Delay Escape* trials (i.e. Negative – Positive feedback in the *Delay Escape* condition) were significantly correlated ($r(32) = .449$). CNV IDES scores (*Delay No-Escape – Delay Escape*) were correlated with inattention, hyperactivity and total ADHD scores across the whole sample ($r(36)=.427$, $p = .009$; see Figure 3), these

correlations were similar for the young adults (r(18) = .406) and adolescent group (r(19) = .391). An association between ADHD scores and the IDII on the LPP in *Delay Escape* trials (i.e. Negative – Positive feedback in the *Delay Escape* condition) did not reach significance (p >.100).

Discussion

Motivated by the delay aversion hypothesis of impulsive choice, the current paper represents the first study to use electrophysiological techniques to examine reinforcing properties of delay in terms of the patterns of cue and feedback-related activation patterns to the opportunity and achievement of escape from delay. We made a number of predictions. First, the prediction that cues signalling the opportunity to escape delay contingent on efficacious task performance would reinforce shorter response times was supported. **While the impact of delay on behaviour can be modelled in terms of the diminishing subjective value of delayed rewards (temporal discounting models) or delay as a barrier to effective self-regulation (executive function models); the current results suggest that delay itself may also represent a potent negative reinforcer and delay escape, a positive reinforcer.**

Second, we predicted that cues signalling the opportunity for delay escape would act as a reinforcer and promote enhanced activations in relation to anticipatory brain potentials as participants, motivated to escape delay, prepared to respond as quickly as possible. In line with this prediction we found enhanced CNV bilaterally to *Delay Escape* compared with *Delay No-Escape* cues. Despite its commonly observed role in effortful preparation (Brunia et al., 2011; Cant & Bickford, 1967; Van Boxtel & Bocker, 2004), there has been less focus on the CNV in relation to reinforcement and no studies relating to negative reinforcers such as delay. Although there is some evidence that the CNV is modulated by positive reinforcers such as monetary reward (Pierson, et al., 1987), more recent work has not replicated this finding (Goldstein, et al., 2006; Goldstein, et al., 2008). The discrepancy between these results might be due to differences between paradigms. For example, Pierson et al. (1987) used a conditioning paradigm in which participants learnt to differentiate between one of three tone stimuli signalling gain, loss and neutral conditions; whilst Goldstein and colleagues (2006) focused on the effect of varying levels of monetary incentive. Goldstein et al. (2006) note that differential sensitivity to reward may arise due to personality trait differences amongst participants and, similarly, Pierson et al. (1987) found a larger effect in hedonic compared with anhedonic participants, suggesting that individual differences in the ability to experience pleasure may influence the sensitivity of the CNV to reinforcement. In the current study, the enhanced CNV response to *Delay Escape* relative to *Delay No-Escape* cues did not differ between time-windows (i.e. CNV1 and CNV2). Although previous work has suggested an alerting role for the early subcomponent of the CNV and a stronger preparatory role for the later subcomponent (Loveless & Sanford, 1974; Van Boxtel & Bocker, 2004), it is possible that the period between the cue and target was not sufficiently long enough to disentangle these two sub-components (more commonly examined with cue-target periods of 3-4 seconds) and instead, these results likely reflect the combination of associated alerting and preparatory processes.

Our third prediction related to the neural response to success or failure in the *Delay Escape* condition. We examined the brain response to failures to escape delay and to successful trials during which the engagement of effortful processes had resulted in the avoidance of delay. The failure to escape delay may be described in everyday terms as disappointment. Here we focused on the LPP as a replicated and robust marker of the emotional and motivational salience of and as a potential neural correlate of disappointment. Once again this prediction was confirmed with enhanced LPP amplitude to failures to escape delay when compared to trials in which delay had successfully been escaped. Considered alongside previous literature demonstrating that the role of LPP is modulated by feedback salience, this result **provides some support for models highlighting the motivational significance of delay aversion and delay escape as a driver of human performance (Sonuga-Barke et al., 2010).** Interestingly,

these ERP effects of condition and/or outcome were observed across electrode sites and were seen in both the first and second time windows of the two components. This finding is consistent with the notion that the LPP represents the sustained processing of inherently motivationally salient stimuli.

The reported effects were observed in both adolescents and young adults and the possible prediction of stronger effects in adolescents were not born out. Adolescent brain development has recently received considerable attention (Somerville & Casey, 2010; Somerville et al., 2010) and fMRI evidence suggests that, in contrast to young adults, adolescents exhibit hyper-responsive activation to rewards (e.g. Casey et. al., 2008; Somerville et. al., 2010), which may be associated with increased reward-seeking and impulsive choice. Strong age-related differences were not identified either in neural or behavioural correlates of the anticipation of delay or imposition of delay in the EDI task. However, due to decisions regarding sampling strategy, gender was not equally distributed in either the adolescent or adult sample, the adolescent sample was entirely male and the adult sample was predominately female. Therefore, our research has not allowed us to investigate the effects of age independently of gender, which may impact on delay aversion (e.g. Paloyelis, Asherson, & Kuntsi, 2009). While the current study provides no evidence for differential effects of delay escape as a reinforcer on the performance and brain potentials of male adolescents and predominately female young adults, future work should replicate these findings in gender-matched samples and larger sample sizes should explore these effects further.

Our final predictions considered the extent to which the neural response to delay and delay escape varied as a function of ADHD symptoms. With regard to the CNV, there was a striking association between the differential effect of *Delay Escape* and *Delay No-Escape* conditions on the CNV and ADHD. This finding suggests that delay escape motivated greater levels of response preparation in those individuals with higher ADHD symptoms. The predicted association between the differential effect of success and failure of *Delay Escape* on the LPP and ADHD did not reach significance. **These results provide some support for fMRI evidence which suggests that ADHD-related atypicalities in the neural response to reinforcement are especially marked to incentive-predicting cues and not reward outcome (e.g. Scheres et al., 2007). Future work however should extend the current findings to clinical samples of ADHD patients.**

This is the first study to explore the neural correlates of motivational salience of delay imposition and escape. Future research can build on the current findings. First, the EDI could be modified to include a range of delay values to better examine parametric effects of delay magnitude on neural responses to delay escape. Do the negative reinforcing effects of delay operate in an all or nothing binary fashion or will individuals work increasingly harder to avoid longer delays? It would also be interesting to include an extra non-contingent *no-delay* condition in which no delay was imposed irrespective of performance. Second, in order to explore the relationship between the motivating effect of delay escape and impulsive choice, a battery of tasks looking at smaller sooner versus larger later choices could be added. This should include both real and hypothetical rewards. Third, the current EEG-based analysis could be supplemented with fMRI in order to localise these neural responses to specific generators. There are two obvious predictions. In the anticipation of immediate reward, the delay aversion hypothesis postulates that for individuals who are especially averse to delay (e.g. patients with ADHD), reward-related brain regions such as the ventral striatum will be hypoactivated and it is a hypofunctioning mesolimbic dopamine system which is argued to give rise to impulsive choice (Sonuga-Barke, 2002; Sonuga-Barke, 2003; Sonuga-Barke, 2005). Consistent with this notion, several recent studies have reported hypoactivation of the ventral striatum during reward anticipation in patients with ADHD (Scheres et al., 2007; Ströhle et al., 2008). Next, this model also suggests that the processing of negative reinforcers such as delay will elicit a negative affective response. Preliminary support for this account comes from a recent fMRI study by Plichta and colleagues (2009), who found enhanced amygdala activation to delayed rewards in patients with ADHD.

Fourth, although we have shown that delay escape motivated greater levels of response preparation in individuals with high ADHD symptoms, we have not compared this response to other incentives, such as monetary incentives, or to punishments such a monetary loss. It will be important for future research to compare CNV response to different incentives (and disincentives) to compare the relative motivational salience of delay. Fifth, the correlation between ADHD and the differential effect of delay escape on CNV amplitude needs to be replicated in a large clinical sample using a better set of clinical measures. Sixth, although age effects were not observed in the current study, this analysis should be extended to a larger sample and greater age range, including childhood and adolescent years. Seventh, the two samples performed slightly different versions of the same task. Namely the delay between the informative cue and imperative stimulus were presented at a fixed interval in the task performed by the adult sample, however this delay was jittered in the adolescent sample (the ISI was varied randomly between 2000 and 2500ms). Although we found that there were no differences in the CNV of the two samples, it is theoretically possible that the jittering (which makes the imperative cue less predictable) may have impacted on the CNV. Future research should replicate these findings.

In summary, using a novel task we demonstrated the reinforcing properties of delay escape at both a behavioural and neural level. More specifically neural markers of motivated attention processes and effortful preparation were enhanced to *Delay Escape* relative to *Delay no-escape* cues and this was related to ADHD symptom scores. Furthermore elevated LPP amplitude in settings where delay escape attempts were not successful further supported the salience of delay. **These findings provide preliminary support for the notion that delay itself is motivationally salient and its effects are not restricted to the value of rewards or its impact on self-regulatory abilities.**

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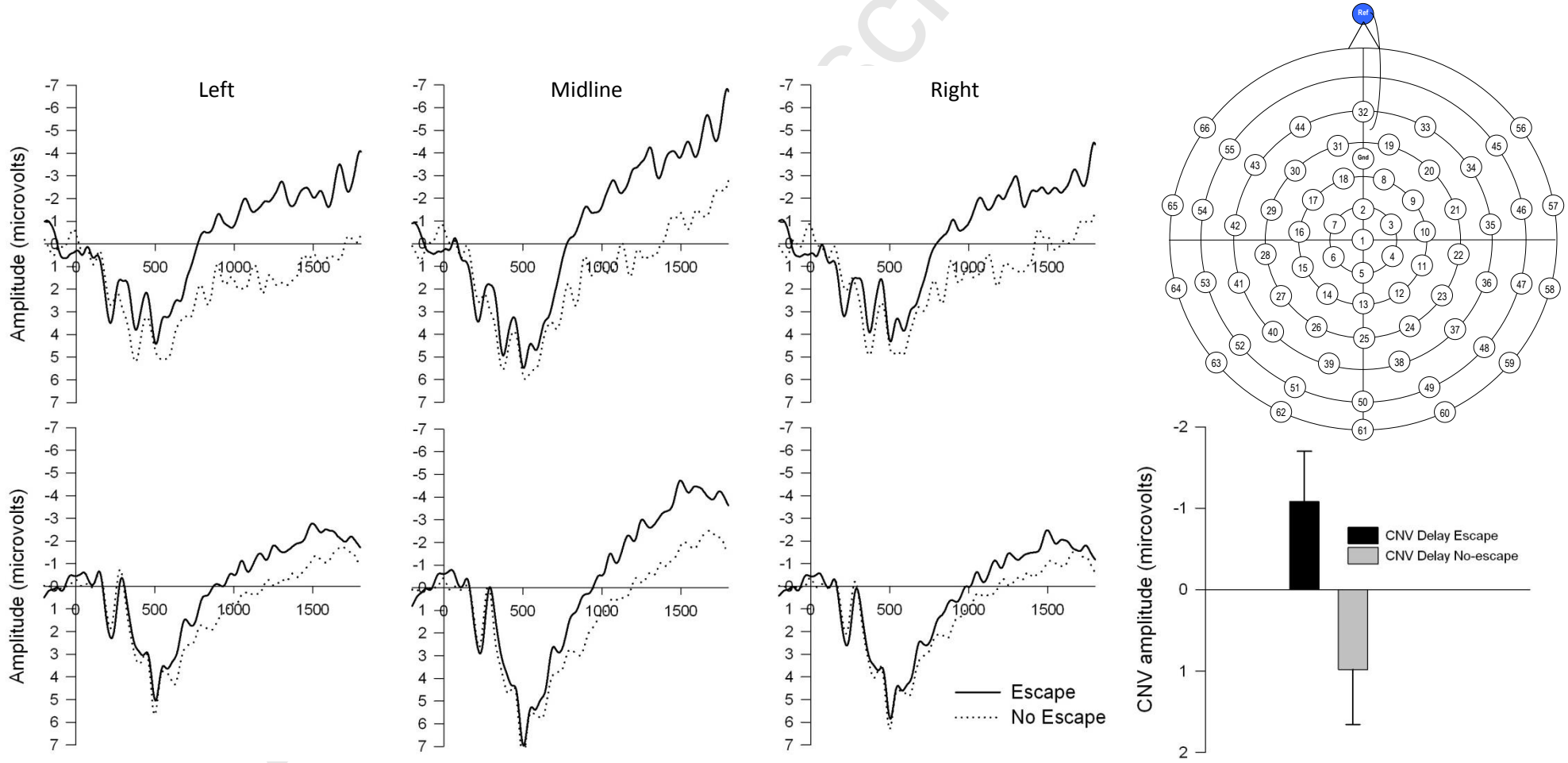

Figure 1. ERP data to *Delay Escape* (solid line) and *Delay No-Escape* (dashed line) cues in left hemisphere, midline and right hemisphere regions for adolescent (above) and young adult (below) samples. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis. On the far right, the bar chart shows the mean CNV condition difference and +/- 1 SE. The equidistant montage is also shown for reference.

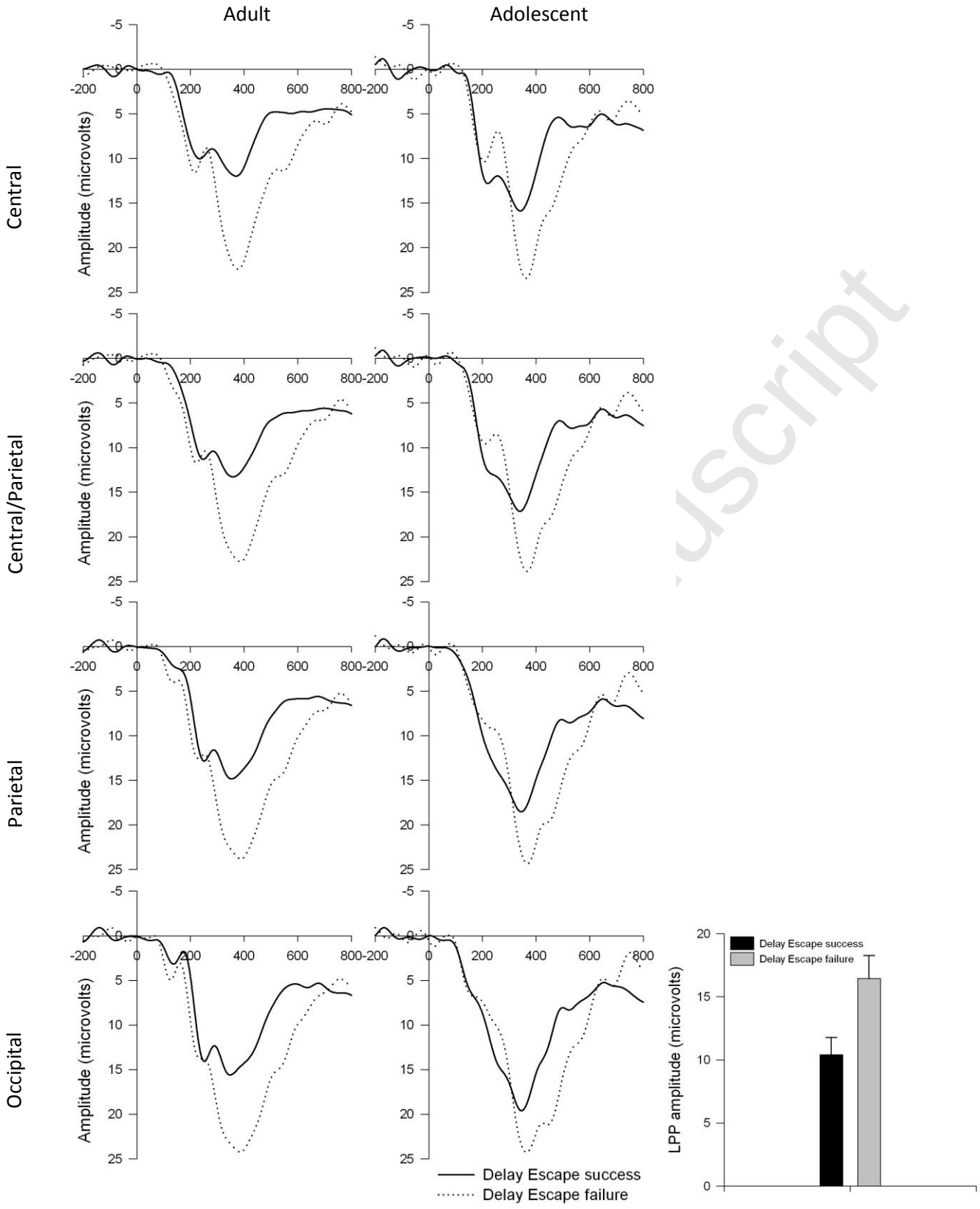

Figure 2. ERP waveforms to feedback indicating success (solid line) and failure (dashed line) to avoid delay in the *Delay Escape* condition in the adult (left) and adolescent (right) samples. Amplitude is shown in μV on the y-axis and time in milliseconds along the x-axis. On the far right, the bar chart shows the mean LPP Delay Escape feedback difference and +/- 1 SE.

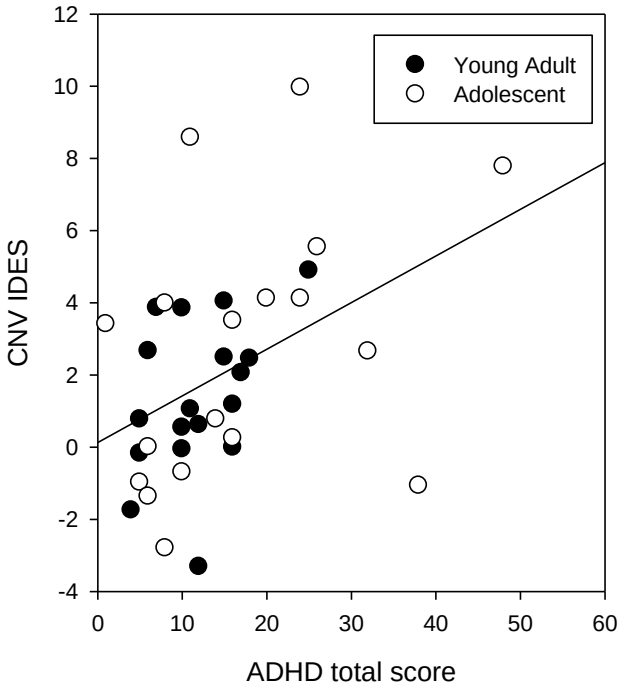

Figure 3. Correlation between ADHD and the Impact of Delay Escape score (*Delay No-Escape – Delay Escape*) for the CNV (r =.427, p = .009).